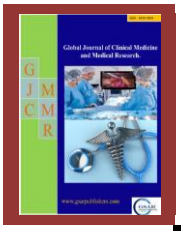


Global Journal of Clinical Medicine and Medical Research [GJCMMR]
ISSN: 2583-987X (Online)
Abbreviated key title: Glob.J.Clinic.Medici.Medica.Res.
Frequency: Monthly
Published By GSAR Publishers
Journal Homepage Link- https://gsarpublishers.com/journal-gjcmmr-home/

POST-MORTEM DIAGNOSIS AND MEDICO-LEGAL ISSUES OF CARDIAC SUBSEQUENT DEATH IN A CONTEXT OF STRESS

(About an autopsy case)

By

Dr. Mohammed Amine DAERQAOUI[1], Dr. Awatif AHERI[2], Dr. Fatima BELAMINE[3], Dr. Ahmed BELHOUSS[4], Dr. Hicham BENYAICH[5]

[1,2,3,4,5]*Department of Forensic Medicine, Ibn Rochd University Hospital Center, Casablanca, Morocco.*

Article History

Received: 22/05/2025
Accepted: 27/05/2025
Published: 29/05/2025

Vol – 3 Issue – 5

PP: -10-21

Abstract

Introduction: *The interaction between stress, whether physical or emotional, and cardiac events is proving critical, especially when no pre-existing heart disease is apparent. This dynamic raises both medical and legal questions, such as how stressful events can lead to fatal outcomes, and how such cases are handled legally. This complexity highlights the need for interdisciplinary approaches to assessing cause and responsibility in such scenarios, incorporating established medico-legal criteria.*

Case report: *The reported case concerns a 64-year-old man, with no notable medical history, who was physically assaulted by his son-in-law. Suffering from chest pain, he consulted the emergency department, where he was diagnosed with chest pain with no abnormalities on chest X-ray. A subsequent altercation led to his sudden death. Autopsy revealed a ruptured left ventricle with significant hemopericardium, with no apparent external traumatic lesions.*

Discussion: *The medical section of the article examines the impact of emotional and physical stress on pre-existing cardiac conditions, highlighting the importance of thorough evaluation in determining cause of death. It also discusses the diagnostic challenges encountered at autopsy and the implications of these findings for forensic evaluations. Legally, the article compares Moroccan legislation concerning death without intent to kill with international standards, explaining the nuances between different jurisdictions. The discussion also incorporates Davis' criteria, essential for assessing responsibility in stress- and violence-related deaths.*

Conclusion: *The need for interdisciplinary collaboration is emphasized to accurately assess the medical causes of death and their legal implications. Emphasis is placed on improving post-mortem diagnostics and strengthening the attribution of legal responsibility for more effective management of medico-legal cases and better understanding by healthcare professionals and judicial authorities.*

Keywords: *Myocardial rupture - Myocardial infarction - Davis criteria - Stress - Criminal responsibility.*

1. Introduction

For a long time, the potentially fatal consequences of emotional stress have been ingrained in popular belief, often evoked by expressions such as ‘broken heart’ and ‘scared to death.’ The importance of this phenomenon is not limited to the medical field. From a legal perspective, cardiac death linked to a stressful event raises significant questions, particularly regarding criminal liability. It poses challenges in cases where the triggering event is associated with external factors such as harassment, verbal assaults, or severe interpersonal conflicts. Consequently, attributing a death to an emotional cause imposes strict requirements in terms of forensic investigations.

Faced with such situations, forensic pathologists, police officers, and prosecutors are often at a loss when it comes to determining the immediate causality of a death following a violent act, especially in the absence of direct physical contact or visible fatal traumatic injuries during the autopsy.

In the Moroccan context, it is crucial to distinguish between the notions of ‘violence without intent to cause death’ and ‘murder,’ terms that can differ significantly from those used in other international legal systems, which more commonly refer to ‘homicide.’ This distinction is essential for assessing criminal liability in cases of sudden death where emotional stress plays a potentially critical role.

*Corresponding Author: **Leila BELAROUSSI**

2. Case report :

A. Circumstances :

The case involves a 64-year-old man, married, father of six children, retired, with no significant medical history, specifically no smoking or alcohol use, no diabetes, no hypercholesterolemia, no hypertension, and no history of heart disease. He was reportedly the victim of physical aggression by his son-in-law, involving kicks to the chest area, after which he experienced chest pain. Following this, he presented to the emergency department of a hospital, where a chest X-ray was ordered, and he received a medical-legal certificate noting pain in the left hemithorax, respiratory discomfort, and left shoulder pain, with a work incapacity duration set at twenty days.

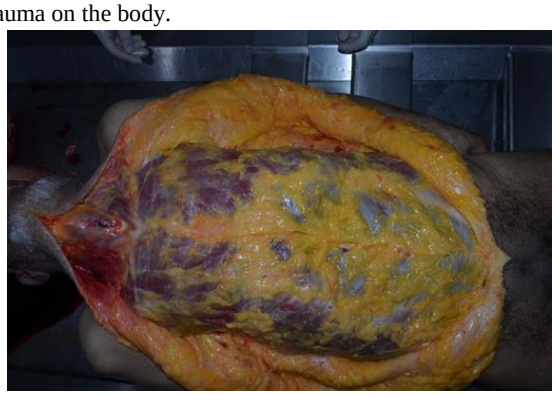
Due to the worsening of his condition, the patient reportedly returned to the same hospital emergency department the next day and was referred to another hospital for evaluation of trauma involving the left thoracic region and left shoulder, following the violent incident. A thoracic surgery consultation was requested, and a chest X-ray was performed, which came back normal.

The patient reportedly returned home, where he had a second altercation with his daughter's husband. After this, he experienced a sudden collapse with loss of consciousness. He was transported by ambulance to a nearby hospital, where he arrived deceased the same day.

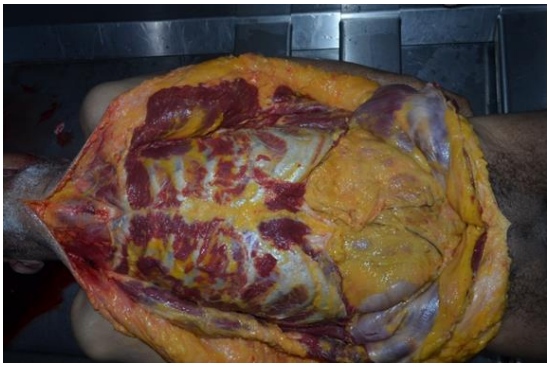
The body was transferred to the Forensic Medicine Department of CHU Ibn Rochd for autopsy.

B. Thanatological data :

The corpse presented to us was that of an adult male, of medium build, with no The corpse presented to us was that of an adult male, of medium build, with no external signs of trauma on the body.



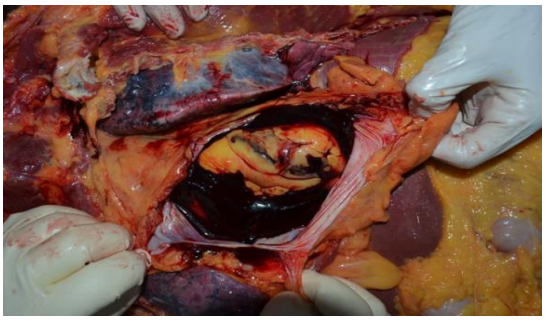
Picture 1 : Absence of hemorrhagic suffusions in the thoracic fatty tissue, and absence of traumatic signs



Picture 2 : Absence of hemorrhagic suffusions on the thoracic cavity.

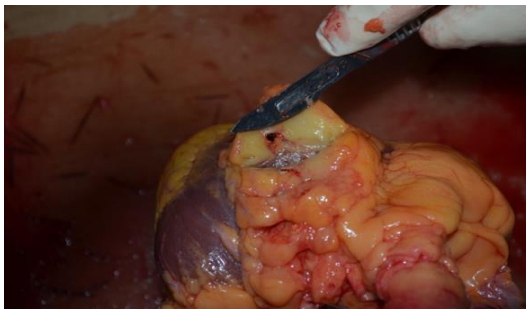
The autopsy examination noted the absence of internal signs of trauma to the cellulo-fatty thoracic tissue (Picture 1) and the absence of trauma to the thoracic muscles (Picture 1) and rib cage (Picture 2) on his body.

Autopsy examination revealed a hemopericardium estimated at 580 ml made up of coagulated blood (Picture 3).



Picture 3 : Hemopericardium made of coagulated blood.

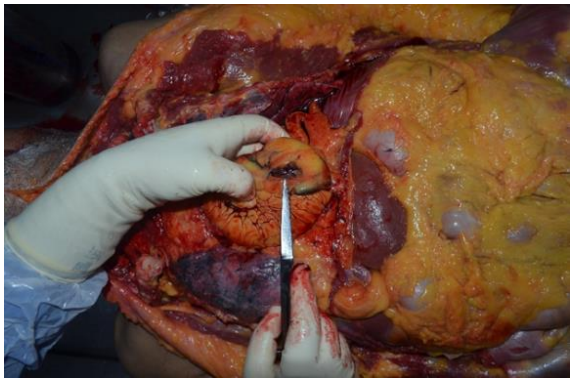
The heart weighed 343 g. Dissection of the coronary arteries revealed 20% eccentric atheromatous thickening in the common trunk, 30% eccentric atheromatous thickening in the proximal third of the anterior interventricular artery, 40% eccentric atheromatous thickening in the proximal third of the anterior interventricular artery, then 40% eccentric atheromatous thickening with a **red thrombus obstructing the lumen starting in the proximal third and extending over four sections to the middle third** (Picture 4), insignificant thickening in the middle third of the circumflex, 20% eccentric thickening in the proximal third of the right coronary artery and 90% eccentric thickening in its middle third.



Picture 4 : Thrombus obstructing the anterior interventricular artery.

*Corresponding Author: **Leila BELAROUSSI**

Examination of the myocardium revealed a **rupture on the anteroinferior surface of the left ventricle** (Picture 5), with haemorrhagic suffusion opposite, and extended to the epicardial fat on the anterior surface of the heart between the two ventricles, associated with local myocardial hyperhaemia and a few areas of whitish fibrosis anteroseptally. Examination of the valvular and subvalvular apparatus was unremarkable.

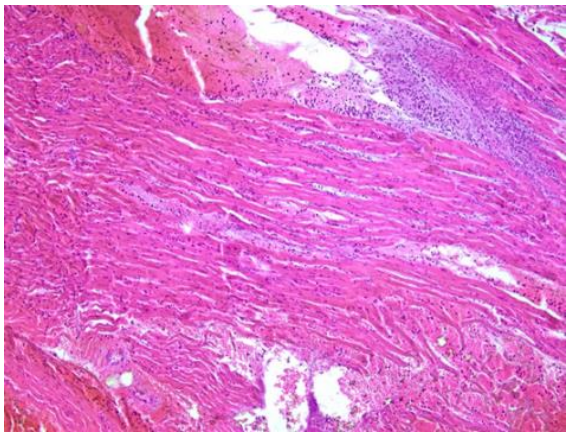


Picture 5 : Trans-mural rupture in the anteroinferior aspect of the left ventricle.

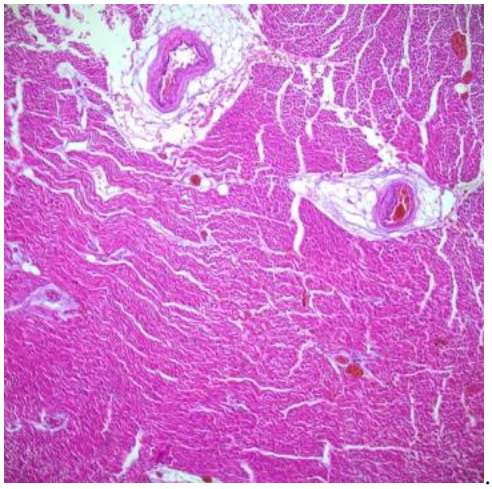
We measured troponin I Hs (a cardiac enzyme whose levels rise in coronary infarction) in vitreous humor and pericardial blood. It was elevated to 20,000 and over 50,000 ng/l respectively (normal range 0-34 ng/l).

Intra-autopsy samples of biological fluids (blood, urine and gastric fluid) were taken. The results were negative.

Histopathological examination showed that the anteroinferior wall of the left ventricle was the site of haemorrhagic remodelling..



Picture 6 : Microscopic image with standard HES stain showing cardiac parenchyma with hemorrhagic changes et inflammatoires (x10)



Picture 7 : Microscopic image with standard HES stain showing congestion within the cardiac parenchyma.

Microscopic examination of the ventricular wall reveals haemorrhagic suffusions, fibrinoid material and sheets of neutrophils (Picture 6 and 7).

A. Cause of death and issues:

We have reported that death was secondary to massive hemopericardial tamponade complicating a left ventricular rupture following an acute myocardial infarction, in a patient with stigmata of chronic ischemic heart disease.

Except that this myocardial rupture occurred in a context of aggression and altercation, several essential questions therefore arise and require in-depth medico-legal analysis. The issues raised can be formulated as follows:

1. *Nature of rupture*: Is it post-traumatic myocardial rupture due to direct impact, or rupture due to spontaneous myocardial ischemia potentially exacerbated by the emotional stress of the altercation?
2. *Evolution of a myocardial infarction:* What is the usual delay between a myocardial infarction and myocardial rupture, and do these delays correspond to the intervals observed between attacks and death?
3. *Post-traumatic survival:* Is it plausible that mechanical myocardial rupture due to physical trauma may not manifest itself fatally until one day after the initial traumatic event?
4. *Contribution of the patient's pre-existing condition:* To what extent might the patient's pre-existing health conditions have influenced the severity of the response to the insult and myocardial ischemia, and would an individual without pre-existing cardiac pathologies have had a different outcome?
5. *Medical responsibility:* Were there any shortcomings in medical care during the first visit to the emergency room that could have contributed to the death, and could this death have been avoided with a different intervention?
6. *Forensic accountability:* Can the myocardial rupture be directly attributed to the first physical

*Corresponding Author: **Leila BELAROUSSI**

assault or the second altercation, and what is the causal link between these events and the death?

7. *Criminal liability of the aggressor:* What should be the legal qualification of the aggressor's action? Is it violence resulting in death without intent, murder, or a lack of criminal responsibility due to the absence of a direct link between the aggression and the death?

3. Discussion :

In order to frame the discussion, we will proceed with a medical and legal discussion:

A. Medical discussion :

1. Pathophysiology of Sudden Cardiac Death and Emotional Stress:

Sudden cardiac death linked to emotional stress is the result of a complex interaction between several pathophysiological factors. Activation of the sympathetic nervous system plays a central role, triggering a cascade of physiological reactions that can lead to a fatal outcome.

Sympathetic Nervous System Activation and Catecholamine Release: Intense emotional stress activates the sympathetic nervous system, leading to a massive release of catecholamines (adrenalin and noradrenalin) by the adrenal glands (1). These hormones trigger a series of physiological responses, including an increase in heart rate, blood pressure and myocardial contractility.

Coronary vasospasm: Increased catecholamine levels can cause coronary vasospasm, a constriction of the coronary arteries that reduces blood flow to the heart muscle (2). This mechanism is often involved in myocardial infarction with unobstructed coronary arteries.

Cardiomyocyte necrosis: Over-stimulation by catecholamines can lead to a direct toxic effect on myocardial cells, i.e. cardiomyocyte necrosis, characterized by contraction band necrosis due to intracellular calcium overload and hypercontractility of myocardial cells (3). This form of myocardial lesion is often observed in the context of acute emotional stress.

Activation of the Renin-Angiotensin-Aldosterone System (RAAS): Emotional stress also activates the RAAS, leading to sodium and water retention, increasing blood pressure and cardiac afterload (4). This further worsens the heart's workload, especially in individuals with already compromised myocardial function.

2. Immediate causes of death :

The immediate causes of death in sudden cardiac death are often related to fatal arrhythmias or acute heart failure. These events can occur in the context of various underlying cardiac pathologies exacerbated by emotional stress.

Fatal arrhythmias: Ventricular arrhythmias such as ventricular fibrillation and ventricular tachycardia are frequent causes of sudden death (5). Emotional stress can increase susceptibility to arrhythmias through the pro-arrhythmic effects of catecholamines.

Acute heart failure: results from the inability of the myocardium to maintain adequate perfusion due to extensive myocardial damage or severe dysfunction (6). This phenomenon is aggravated by emotional stress, which increases myocardial metabolic demand.

3. Underlying cardiac pathologies :

Underlying cardiac pathologies play an important role in predisposing individuals to the lethal effects of emotional stress. Here is how these pathologies interact with the described pathophysiological mechanisms.

Coronary artery disease: Patients with coronary artery disease have atherosclerotic plaques that can trigger ischemic episodes in response to emotional stress, increasing the risk of sudden death (7).

Individuals with underlying coronary atherosclerosis are particularly vulnerable to the effects of emotional stress. The atheromatous plaque may rupture, causing a myocardial infarction (8).

Cardiomyopathies: Certain cardiomyopathies, such as hypertrophic cardiomyopathy and dilated cardiomyopathy, can predispose to fatal cardiac events under stress. These conditions increase myocardial susceptibility to stress-induced ischemia and arrhythmias (9).

Vasospasm syndromes: Patients with coronary vasospasm syndromes, such as Prinzmetal's angina, are also at increased risk of sudden death in response to emotional stress. These syndromes are characterized by transient spasm of the coronary arteries, which can lead to acute myocardial ischemia (10).

Stress cardiomyopathy: Stress cardiomyopathy, also known as Takotsubo syndrome, is characterized by transient left ventricular dysfunction often triggered by emotional or physical stress (11). Clinically, it mimics a myocardial infarction.

4. Diagnostic challenges :

Differentiating between causes of sudden death linked to emotional stress and other etiologies requires a rigorous, methodical approach. This includes toxicological and genetic analyses where possible. Identifying specific cardiac lesions is crucial to establishing the precise cause of death and for medico-legal reasons, such as assessing the responsibility of the aggressors and providing accurate forensic expertise.

a- Identification of lesions :

It is sometimes difficult to identify cardiac lesions on macroscopic examination. Visible signs such as myocardial hypertrophy or scars from previous infarctions may be absent or subtle. The presence of coronary thrombi or plaque ruptures may also go unnoticed without careful dissection of the coronary arteries. Sometimes, even obvious macroscopic abnormalities can be missed due to post-mortem decomposition or inappropriate autopsy techniques (12).

Histopathological analyses play an important role in identifying microscopic lesions, but they are not without their challenges. Histopathological signs of sudden cardiac death,

*Corresponding Author: **Leila BELAROUSSI**

such as contraction bands and cardiomyocyte necrosis, can be difficult to interpret. These lesions can sometimes be confused with post-mortem artifacts or non-specific changes. In addition, the distribution and extent of lesions may be patchy, requiring extensive sampling for accurate assessment (13). Post-mortem laboratory tests, including assays for cardiac enzymes such as troponin, can help confirm acute myocardial ischemia. However, the levels of these biomarkers can be influenced by various post-mortem factors, making their interpretation tricky (14).

b- Differentiating between post-traumatic and spontaneous MI :

 # Detailed description of pathophysiology

Chest trauma can induce myocardial infarction (MI) through several complex pathophysiological mechanisms. Firstly, the direct force of the impact can cause coronary artery spasm, which is a sudden and severe constriction of vascular smooth muscle. This spasm can temporarily interrupt the blood supply to the heart muscle, leading to ischemia and potentially infarction (15).

In addition, the intense physiological and emotional stress associated with trauma can cause an acute rise in circulating catecholamines, exacerbating arterial spasm and increasing myocardial oxygen demand, which can precipitate rupture of pre-existing atherosclerotic plaque. This rupture can then lead to the rapid formation of an occlusive thrombus in the affected coronary artery, blocking the blood supply and causing MI (16).

 # Elements of differential diagnosis

The differential diagnosis between myocardial contusion and myocardial infarction in patients with thoracic trauma is complex. Cardiac magnetic resonance imaging (MRI) plays a vital role in this context, as it can specifically detect the myocardial edema and areas of necrosis characteristic of myocardial infarction, even in the absence of significant coronary disease. It also visualizes the presence of intramyocardial bleeding, typical of contusion. (17). However, the limited accessibility of MRI and potential contraindications often necessitate the use of other imaging modalities.

Echocardiography, particularly in transthoracic and transesophageal modes, is another valuable modality. It enables rapid assessment of cardiac function, detection of segmental motility abnormalities, pericardial effusions and other post-traumatic structural abnormalities (18). For patients in whom MRI is not feasible, myocardial scintigraphy may also be considered to assess myocardial perfusion and viability (19).

Cardiac computed tomography (CT), although less sensitive than MRI for detecting specific myocardial lesions, may be useful for assessing other associated thoracic injuries, such as bone fractures or large vessel lesions (20).

N G D Laguérenne et al (2007) examined a rare case of myocardial infarction (MI) following closed chest trauma in a 58-year-old man. This case illustrates how a car accident with direct impact on the thorax could induce myocardial infarction without prior triggering of atherosclerosis or other common coronary pathologies.

 # Traumatic injuries considered:

1. **Myocardial contusion:** the patient suffered direct contusions to the thorax, manifested by bruising of the skin opposite the seat belt and rib fractures, which could have transmitted forces directly to the heart, resulting in myocardial damage.
2. **First diagonal artery lesions:** Although coronary angiography revealed no significant coronary lesions, it did show a non-significant plaque with an inhomogeneous appearance on the proximal portion of the first diagonal artery. This observation suggests that the trauma may have caused a specific vascular lesion that did not result in significant obstruction but was sufficient to disrupt myocardial function.
3. **Rib fractures and pulmonary contusions:** In addition to fractures of the first to fourth left ribs, the patient presented with a right lower pulmonary contusion. This damage indicates significant force transmission to the thorax, which may also affect the underlying heart.
4. **Disturbances in segmental kinetics:** echocardiography revealed anteroapical akinesia, indicating altered motility in this region of the heart, presumably in response to the traumatic injury.
5. **Electrical disturbances:** The patient's electrocardiogram on admission showed abnormalities characteristic of myocardial damage, such as ST-segment elevations, which are typical of acute cardiac injury (21).

Aya TAKADA et al (2006) conducted a study of left ventricular ruptures occurring during acute myocardial infarction, leading to sudden out-of-hospital death. The study examined 50 cases to assess endocardial tears and rupture pathways through macroscopic and microscopic examinations. To differentiate post-traumatic myocardial infarction (MI) from MI of non-traumatic origin, several elements must be taken into account, mainly based on pathological features and clinical circumstances.

Here are the main differentiating factors, supported by information provided in the article "Endocardial tears and rupture tracts of left ventricular ruptures during acute myocardial infarction (22)":

1. **Clinical history:**

- Post-traumatic MI: often preceded by direct trauma to the thorax, as in car accidents or direct impact injuries.
- Non-traumatic MI: generally associated with cardiovascular risk factors such as hypertension, hyperlipidemia, diabetes or smoking.

2. **Location and nature of lesions:**

- Post-traumatic MI: may present myocardial tears or ruptures at atypical sites that do not correspond to the supply territories of the main coronary arteries.

*Corresponding Author: **Leila BELAROUSSI**

- Non-traumatic MI: ruptures or tears are generally located in areas of recent infarction where the tissue is weakened, often in relation to the supply territories of the occluded coronary arteries.

3. Pathological examination of thrombi:

- Post-traumatic MI: The presence of thrombi may be less frequent or atypical.
- Non-traumatic MI: Mature thrombi are frequently present in ruptures, indicating a more gradual progression of the lesion.

4. Characteristics of myocardial rupture:

- Post-traumatic MI: Ruptures may occur more abruptly and are often associated with more extensive physical damage due to the force of the impact.
- Non-traumatic MI: Tears and ruptures are often the result of extensive necrosis of myocardial tissue, progressing over several days after coronary occlusion.

5. Macroscopic and microscopic examination:

- Post-traumatic MI: examination may reveal signs of contusion, hemorrhage or impact injury.
- Non-traumatic MI: typically shows signs of ischemia and necrosis associated with specific areas of infarction without external signs of direct trauma.

In summary, the distinction between a post-traumatic MI and a MI of non-traumatic origin is based on a detailed assessment of the clinical history, the morphological characteristics of the cardiac lesions, and the results of pathological examinations.

 # Other differential diagnoses:

To distinguish sudden death related to emotional stress from other causes, it is essential to consider a wide range of etiologies:

1. Drug poisoning and toxic substances:

- Illicit drugs: Cocaine, amphetamines, opioids (23, 24,25).
- Prescription drugs: Tricyclic antidepressants, beta-blockers, antiarrhythmics (26).
- Legal substances: Alcohol, nicotine (27).

2. Congenital Heart Defects:

- Hereditary cardiomyopathies: hypertrophic cardiomyopathy, dilated cardiomyopathy (28).
- Ion channel abnormalities: Long QT syndrome, Brugada syndrome (29).
- Structural malformations: Tetralogy of Fallot, Ebstein's anomaly (30,31).

3. Acquired heart disease:

- Coronary heart disease: myocardial infarction, stable and unstable angina pectoris (32).
- Heart failure: acute and chronic (33).
- Valvulopathies: aortic stenosis, mitral regurgitation (34).

4. Other pathological conditions:

- Infections: Myocarditis, endocarditis (35).
- Metabolic disorders: Hyperthyroidism, hypokalemia (36).
- Neurological causes: stroke, epilepsy (37).

B. Legal discussion:

1. Analysis of Moroccan legislation :

Every offence is made up of three main elements: the legal element, the material element and the moral element. For this offence to be established, it is necessary to demonstrate the existence of these three elements.

a. Legal element:

This is the legal basis for the offence. For conduct to be considered an offence, it must be provided for and punished by law. In other words, there must be a legal text (a provision of the Penal Code, for example) which defines the behavior as an offence and sets out the applicable penalties.

Article 403 of the Moroccan Penal Code stipulates that: "*When wounds or blows or other violence or assaults committed voluntarily but without the intention of causing death have nevertheless caused it, the penalty is ten to twenty years' imprisonment. Where there has been premeditation or an ambush or the use of a weapon, the penalty is life imprisonment.*"

This article is fundamental to dealing with cases where violence, even without murderous intent, leads to the victim's death. It provides a legal framework for situations where intense physical or emotional aggression leads to death without intent to kill.

b. Material element:

- Criminal activity:

The incriminated activity includes any form of violence or assault resulting in bodily harm or damage to the victim's health. The material element also includes the circumstances and consequences of the act. This includes actions such as tying the victim up with a rope, or threatening them with a weapon to terrorize them..

- **Results :**

The victim's death is the result of the offence. In the event of death following an assault, the criminal offence is classified as "intentional violence resulting in death without intent to cause", punishable by 10 to 20 years' imprisonment. If the act is premeditated, it is classified as simple murder, punishable by life imprisonment.

- **Causal link:**

A causal relationship must be established between the attack and the death. The role of the forensic pathologist is crucial in confirming this link through forensic expertise..

c. Moral element:

The moral element is based on the intention to cause harm. Even if the intention to kill is not present, the intention to cause harm is sufficient. What matters is the result: if the violence leads to death, the perpetrator is held responsible for this consequence.

The legal qualification in the reported case suggests several possibilities, whether the assailant had no explicit intention to kill, the action was probably intended to intimidate, threaten or cause harm, but with no intention to cause death, or there was a premeditated intention to cause death. Therefore, if the death was attributed to the alleged aggressor, the qualification

*Corresponding Author: **Leila BELAROUSSI**

of "violence without intent to cause death" is more appropriate than "murder".

The concept of "violence without intention to cause death" used in Morocco could be similar to various legal qualifications in other legal systems, depending on the context of the act and its consequences. For example:

- France: The term "coup et blessures ayant entraîné la mort sans intention de la donner" is used in article 222-7 of the French Penal Code, which seems to be the most direct equivalent of Moroccan terminology.
- United States: The closest term would probably be "involuntary manslaughter", specifically in cases where the intent to kill is not present, but death nevertheless results from criminal negligence or a non-lethal illegal act. The latter seems close to the notion of violence without intent to cause death.
- United Kingdom: The corresponding offence could be "Manslaughter by gross negligence" or "Unlawful act manslaughter", depending on whether the death was due to gross negligence or an unlawful act resulting in death.

These distinctions are essential for international comparisons, as they affect the classification of crimes and the corresponding penalties..

2. Case discussion:

1. Nature of rupture: Is it post-traumatic myocardial rupture due to direct impact, or rupture due to spontaneous myocardial ischemia potentially exacerbated by the emotional stress of the altercation?

Distinguishing between post-traumatic and non-traumatic ischemic myocardial rupture in the context of myocardial infarction (MI) requires careful assessment of the patient's cardiac lesions, clinical circumstances and medical history. Here's a detailed analysis based on the case presented :

Typically, myocardial rupture due to direct trauma would require a significant impact observable during post-mortem examination, such as signs of direct contusion or impact lesions (Fracture/ Ecchymosis...). However, in the case presented, although there was an initial thoracic impact, internal and external examination revealed no obvious signs of trauma, which could complicate direct attribution to trauma.

Characteristics of rupture due to ischemia include the presence of areas of fibrosis, hemorrhagic changes and thrombi in the coronary arteries. These features are visible in the dissection description, where a large red thrombus was found in the anterior interventricular artery, covering a large segment and associated with myocardial rupture.

Signs of thrombosis and notable atherosclerotic changes in the coronary arteries raise the possibility that the rupture is linked to an exacerbation of myocardial ischemia.

Significant elevations of troponin I Hs levels in vitreous humor and pericardial blood indicate recent myocardial necrosis, reinforcing the hypothesis of Myocardial Infarction with mechanical complications such as ventricular wall rupture. These elevated biomarkers, combined with signs of rupture in the territory vascularized by the occluded artery, strongly support an ischemic genesis of the rupture.

In summary, although the initial trauma may have played a role in exacerbating pre-existing conditions, Examination of the coronary arteries and myocardium revealed atheromatous thickenings and an obstructing thrombus in the anterior interventricular artery, the location corresponding to the area of myocardial rupture suggest that the rupture may have been primarily due to myocardial infarction exacerbated by the stressful conditions experienced by the patient.

2. Evolution of a myocardial infarction: What is the usual delay between a myocardial infarction and myocardial rupture, and do these delays correspond to the intervals observed between attacks and death?

Myocardial rupture is a serious but rare complication of myocardial infarction. It occurs when the heart wall, weakened by the necrosis of myocardial tissue following an infarction, tears, with potentially fatal consequences.

Batts et al. (1990) found that 58% of ruptures occurred within the first five days after myocardial infarction(38). Shapira et al. (1987) reported that the majority (95%) of cardiac ruptures occur within the first six days following acute myocardial infarction, with 40% occurring within the first 24 hours (39). Lewis et al. (1969) found that 69% of cardiac ruptures occurred within the first week after infarction, with an average of five days (40).

Tao et al (2004) found that matrix metalloproteinase (MMP-2 and MMP-9) activity and macrophage infiltration increased in the first few days after infarction, contributing to myocardial rupture due to excessive degradation of the extracellular matrix(41).

Although the stress of the second altercation could precipitate the breakup. The onset of chest pain immediately after the initial attack, documented by the emergency room visit and a forensic certificate, could indicate that the ischemia was already evolving. This is in line with the literature, which states that post-infarction myocardial rupture most often occurs between the first and fifth day after the initial event.

3. Post-traumatic survival: Is it plausible that mechanical myocardial rupture due to physical trauma may not manifest itself fatally until one day after the initial event?

The patient reported chest pains immediately after the first physical assault. He was then seen by a doctor and examined by a thoracic surgeon. His chest X-ray came back normal, and he was then seen by a third doctor, who issued him with a medical-legal certificate.

A direct blow to the chest can theoretically be responsible for atherosclerotic plaque cracking in a patient with advanced

*Corresponding Author: **Leila BELAROUSSI**

coronary atherosclerosis. However, there are no signs of trauma in the thorax or elsewhere, either on external examination or at autopsy..

If the myocardial rupture had resulted from the blow inflicted during the initial physical assault (even though we found no traumatic signs), death would have occurred immediately due to acute cardiac failure and cardiogenic shock (42).

But if the blow had caused initial minor lesions, such as contusions, which would have evolved into rupture and tamponade by the pericardium, the survival time could be a few hours to a day, which is less likely given the extent of the lesions found at autopsy testifying to the severity of the rupture and the patient's previous condition meaning that these compensatory mechanisms would not have allowed survival, and if this were the case, the patient's clinical condition would be alarming. He would have presented symptoms of marked cardiac distress, including intense chest pain and signs of cardiac shock, which should be identifiable by any physician, making it unlikely that such a patient would be able to continue his normal activities or go for consultation or certification without detection of severe symptoms.

4. **Contribution of the patient's previous condition : To what extent might the patient's pre-existing health conditions have influenced the severity of the response to aggression and myocardial ischemia, and would an individual without pre-existing cardiac pathologies have had a different outcome ?**

In a patient with pre-existing cardiac pathologies, a direct physical assault on the thoracic region could precipitate myocardial rupture. Cardiac tissues that are weakened or already damaged by cardiac pathologies (such as atherosclerosis) are less able to withstand physical impacts, which can lead to rupture even with a trauma considered moderate compared to a healthy person. An individual without pre-existing heart disease could better withstand direct thoracic trauma without suffering myocardial rupture. Healthy cardiac tissue has better structural integrity and can tolerate greater forces before rupture.

Patients with pre-existing cardiac pathologies are also at increased risk of developing myocardial infarction in response to acute stress. Stress can exacerbate the underlying conditions causing acute myocardial ischemia. Although less likely to suffer a myocardial infarction under stress than those with pre-existing pathologies, healthy individuals are not entirely immune.

Extreme stress can still trigger a cardiac event through mechanisms such as sympathetic hyperactivation and electrolyte imbalance, but survival rates and clinical outcomes can be significantly better in the absence of underlying cardiac disease.

5. **Medical responsibility: Were there any shortcomings in medical care during the first visit to the emergency room that could have contributed to the death, and could this death have been avoided with a different intervention?**

The issue of medical responsibility in the management of the case after the initial assault raises fundamental questions. If medical responsibility, such as diagnostic error or delay in the initial emergency management, is proven, it could have contributed critically to the fatal outcome. A thorough evaluation of the medical actions, including the clinical examination, the diagnoses evoked, the tests ordered or not (an 'electrocardiogram, for example?), the treatments prescribed or omitted, and the patient's monitoring, would be essential to determine whether there had been a breach.

In the event of post-traumatic myocardial rupture, early detection and emergency surgery would be necessary to avoid a fatal outcome. And in the scenario where rupture resulted from myocardial infarction, exacerbated by emotional or physical stress, early identification of signs of myocardial ischemia and rapid intervention could have been decisive..

Could death have been avoided with a different intervention? The question of whether death could have been avoided with a different medical intervention is crucial in determining medico-legal liability. This analysis must be based on a rigorous evaluation of the care provided, comparing it with recognized medical standards. In particular, it would be relevant to consider whether the performance of an electrocardiogram (ECG) could have influenced the outcome.

To do this, it is essential to have access to the patient's complete medical file and the testimonies of the doctors and medical institution involved, in order to fully understand the decisions made and their justifications..

6. **Medico-legal imputability: Can the myocardial rupture be directly attributed to the first physical aggression or to the second altercation, and what is the causal link between these events and the death??**

Hemorrhagic suffusions and sheets of neutrophils confirm inflammation and an active immune response in the heart, consistent with recent myocardial injury. This can help confirm that the heart has undergone significant stress or direct injury shortly before death.

The absence of overt traumatic signs at autopsy, combined with the chronology of events reported, suggests that the myocardial rupture was more likely due to an infarction exacerbated by the stress of the circumstances rather than direct trauma. Although the second altercation may have increased the stress on the patient, it is likely that ischemia was already in progress.

It is important to note that, in this specific case, both the initial assault and the subsequent altercation are attributed to the same alleged aggressor. This situation raises major issues of legal imputability to a single event, since the same aggressor is involved on two occasions. This singularity simplifies the attribution of responsibility, which would be far more complex if two different aggressors were involved.

*Corresponding Author: **Leila BELAROUSSI**

7. **Criminal liability of the aggressor: What should be the legal qualification of the aggressor's action? Is it violence resulting in death without intent, murder, or a lack of criminal responsibility due to the absence of a direct link between the aggression and the death ?**

There are certain situations where determining the circumstances of sudden cardiac death following a state of emotional stress remains complicated and controversial. In 1978, an American physician, Dr. J.H. Davis, proposed criteria that would have to be met in order to criminally implicate the aggressor. These criteria serve as a guideline for the forensic evaluation of medical examiners in such situations. Initially, there were 4 criteria, covering both the circumstances surrounding the death and the forensic findings:

1- The act of aggression must be of such severity and must contain sufficient intentional elements to kill, injure or frighten, so as to qualify as homicide if physical injury results.
2- The victim must have experienced a serious threat to his or her physical integrity..
3- The circumstances of the attack must be such as to be commonly accepted as highly emotional.
4- Symptoms of cardiac origin must have occurred during the period of response to emotional stress, even if the aggression had ceased.

In applying these criteria to this case, it should be noted that the last criterion is present insofar as the chest pains experienced by the deceased following the attack were indeed of cardiac origin, but were mistaken for post-traumatic chest pain. However, we are unable to comment on the existence of the first 3 criteria, given the contradictory nature of the testimony given in the police report. The reality of chest trauma has not been documented, even though it was mentioned in the medical certificate. This is because the pain associated with this trauma was in fact the pain of a myocardial infarction, and no objective lesion related to this trauma could be identified, either in the medical certificate, or on the chest X-ray, let alone at the autopsy.

These criteria were first discussed in 1995 by Dr. Bernard Knight, in particular in his book Knight's Forensic Pathology. Knight introduced important nuances to take account of medical advances and clinical observations that had emerged since Davis' criteria were first proposed. For example, he stressed the need to take into account pre-existing heart disease, which can predispose a person to a fatal outcome in the event of acute stress, even in the absence of extreme physical violence.

Then, in 2004, they were slightly modified to include cases where an injury had occurred, and to incorporate the National Association of Medical Examiners (NAME) guidelines (Guide for Manner of Death Classification). These criteria are used to guide medical examiners in determining the circumstances surrounding sudden cardiac deaths, and to assess legal liability in cases where emotional stress induced by criminal action may have played a role in death.

In the context of this case, taking into account the Davis criteria and their appropriate interpretation, it is possible to consider the aggressor as potentially responsible for the death under the principle of "violence having led to death without intention to give it". This legal framework is distinct from any medical liability, and focuses on assessing the intent and consequences of the aggressor's actions. It recognizes that, although the intent to kill is not established, the aggressor's actions have aggravated a pre-existing medical condition, thus contributing to a fatal outcome.

It is important to emphasize that, although these criteria provide a basis for assessing responsibility, the final assessment of the facts and the attribution of responsibility remain the responsibility of the judge. This distinction makes it clear that, despite forensic analysis and circumstantial evidence, it is the judicial system that determines the presence or absence of criminal responsibility on the part of the aggressor. And this is the case even in other countries, such as the example reported by the Italians F. D. GIOGIO et al (43).

4 Recommendations for future research and practice:

1. Standardized Protocol for Forensic Autopsy in Suspected Sudden Cardiac Death:

- Pre-autopsy: Detailed collection of circumstances of death and medical history.
- Macroscopic examination: Close inspection of the heart for structural abnormalities or signs of trauma.
- Histopathology: systematic sampling of cardiac tissue for microscopic analysis, targeting signs of myocardial necrosis or ischemia.
- Toxicology: Analyses to exclude the presence of substances that can induce or contribute to cardiac death.
- Genetic examination: Search for mutations associated with hereditary cardiac pathologies that can predispose to fatal cardiac events.

2. Advanced Research on Biomarkers of Cardiac Stress:

- Develop studies to validate biomarkers such as plasma catecholamines, troponin, and natriuretic peptide, specifically in post-stress contexts.

3. Training and Awareness:

- Create continuing education modules for forensic pathologists and investigators on advanced techniques for detecting the causes of sudden cardiac death.
- Raising awareness of the importance of medical details and circumstances of death in forensic analysis.

These recommendations aim to improve diagnostic accuracy and ensure a more standardized and uniform approach to the forensic analysis of suspicious deaths. By adopting a clear protocol and furthering research into biological indicators of cardiac stress, it will be possible to better understand and address the legal and medical implications of sudden death in the context of emotional stress..

*Corresponding Author: **Leila BELAROUSSI**

5. Conclusion

This study highlights the complexity of forensic interpretation of cardiac deaths in acute stress settings. It highlights the need for an interdisciplinary approach to correctly assess the medical causes of death and the resulting legal implications. By gathering detailed medical data and analyzing them through the prism of legal requirements, this research enriches our understanding of the dynamics at work in cases of stress-related sudden death.

The aim is twofold: to improve the accuracy of post-mortem diagnoses and to strengthen the basis for legal liability. Through this approach, we hope to contribute to better management of medico-legal cases, and to raising awareness of these complex issues among healthcare professionals and legal authorities. This reflection is intended as a catalyst for wider discussions on best practice and policy in the treatment of sudden death under stress, encouraging an evolution in both medical and legal professional standards.

6. Reference

1. A Narang et al (2018) A New Educational Framework to Improve Lifelong Learning for Cardiologist, Journal of the American College of Cardiology VOL 71 (4) 454–462.https://www.jacc.org/doi/full/10.1016/j.jacc.2017.11.045
2. Lanza, G. A., et al. (2011). Coronary artery spasm: A 2009 update. International journal of medical sciences 2014, Vol. 11
3. Wittstein, I. S., et al. (2005). Neurohumoral Features of Myocardial Stunning Due to Sudden Emotional Stress. New England Journal of Medicine, Vol 352 (6). , 539-548. https://www.nejm.org/doi/full/10.1056/NEJMoa043046)
4. Esler, M., et al. (2001). "The sympathetic nervous system and hypertension." American Journal of Hypertension, Vol 43 (2): 139S-146S.
5. Zipes, D. P., & Wellens, H. J. J. (1998). Sudden cardiac death. Circulation, 98(21), 2334-2351. https://www.ahajournals.org/doi/10.1161/01.CIR.98.21.2334
6. Jessup, M., & Brozena, S. (2003). Heart failure. New England Journal of Medicine, 348(20), 2007-2018. https://www.nejm.org/doi/full/10.1056/NEJMra021498
7. Falk, E., et al. (2013). Coronary plaque disruption. Circulation Research, 113(12), 1493-1505.
8. Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). "Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy." Circulation, 99(16), 2192-2217 https://www.ahajournals.org/doi/10.1161/01.CIR.99.16.2192
9. Maron, B. J., Gardin, J. M., Flack, J. M., Gidding, S. S., Kurosaki, T. T., & Bild, D. E. (1995). "Prevalence of hypertrophic cardiomyopathy in a general population of young adults." Circulation, 92(4), 785-789. https://www.ahajournals.org/doi/10.1161/01.CIR.92.4.785
10. Maseri, A., et al. (1977). "Coronary vasospasm as a possible cause of myocardial infarction." New England Journal of Medicine, 296(14), 830-834.
11. https://www.nejm.org/doi/full/10.1056/NEJM197812072992303#:~:text=34–38-,Conclusions,not%20represent%20a%20selected%20group.
12. Prasad, A., et al. (2008). Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): A mimic of acute myocardial infarction. American Heart Journal, 155(3), 408-417. https://pubmed.ncbi.nlm.nih.gov/18294473/
13. Basso, C., Aguilera, B., Banner, J., Cohle, S., d'Amati, G., de Gouveia, R. H., … & Thiene, G. (2009). "Guidelines for autopsy investigation of sudden cardiac death." Virchows Archiv, 454(1), 11-18.
14. Lampert, R., et al. (2002). "Emotional and physical precipitants of ventricular arrhythmia." Circulation, 106(14), 1800-1805. https://pubmed.ncbi.nlm.nih.gov/12356633/
15. B Madea, & F Musshoff (2006). "Post-mortem biochemistry." Forensic Science International Volume 165, Issues 2–3.
16. Patel, K. M., Kumar, N. S., Desai, R. G., Mitrev, L., Trivedi, K., Krishnan, S. "Blunt Trauma to the Heart: A Review of Pathophysiology and Current Management." Journal of Cardiothoracic and Vascular Anesthesia, 2021. https://pubmed.ncbi.nlm.nih.gov/34840072/
17. Ibanez, B., James, S., Agewall, S., Antunes, M. J., Bucciarelli-Ducci, C., Bueno, H., … & Vranckx, P. "2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation." European Heart Journal, 2018. https://pubmed.ncbi.nlm.nih.gov/28886621/
18. Kim RJ et al. Relationship of MRI delayed contrast enhancement to irreversible injury, infarct age, and contractile function. Circulation. 1999 Nov 9;100(19):1992-2002. doi: 10.1161/01.cir.100.19.1992. PMID: 10556226. https://pubmed.ncbi.nlm.nih.gov/10556226/
19. Otto, Textbook of Clinical Echocardiography, 5th ed., Elsevier, 2013
20. Iskandrian et Garcia, Nuclear Cardiac Imaging: Principles and Applications, 5th ed., Oxford University Press, 2016
21. Raff GL et al., SCCT guidelines for the interpretation and reporting of coronary computed tomographic angiography, Journal of Cardiovascular Computed Tomography, 2009; DOI: 10.1016/j.jcct.2009.01.001). https://pubmed.ncbi.nlm.nih.gov/19272853/

*Corresponding Author: **Leila BELAROUSSI**

22. N G D Laguérenne et al Infarctus du myocarde secondaire à un traumatisme fermé du thorax : à propos d'un cas Annales de Cardiologie et d'Angéiologie 56 (2007) 211–215
23. Takada A, Saito K, Murai T, Hamamatsu A. Endocardial tears and rupture tracts of left ventricular ruptures during acute myocardial infarction: an autopsy study of 50 out-of-hospital sudden death cases. Pathol Res Pract. 2006;202(12):857-62. doi: 10.1016/j.prp.2006.08.005. Epub 2006 Oct 11. PMID: 17045419.
24. L. B. E. Shields et al, Sudden Death Due To Acute Cocaine Toxicity—Excited Delirium in a Body Packer. Journal of Forensic Sciences Volume 60, Issue 6 p. 1647-1651https://onlinelibrary.wiley.com/doi/10.1111/1556-4029.12860
25. Richards, J.R., et al. Methamphetamine abuse and emergency department utilization, western journal od medicine 1999; 170(4): 198–202. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1305548/
26. A. El Sherbini et al, Opioids-induced inhibition of HERG ion channels and sudden cardiac death, a systematic review of current literature. Trends Cardiovasc Med 2024 Jul;34(5):279-285.https://pubmed.ncbi.nlm.nih.gov/37015297/
27. Rea, T. D., et al. (2005). "Incidence of out-of-hospital cardiac arrest.
28. Thun, M. J., et al. (1997). "Alcohol consumption and mortality among middle-aged and elderly U.S. adults." New England Journal of Medicine, 337(24), 1705-1714. https://www.nejm.org/doi/full/10.1056/NEJM199712113372401
29. Maron, B. J., et al. (2013). "Hypertrophic cardiomyopathy." The lancet, Vol 381, issue 9862. https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(12)60397-3/abstract
30. A A M Wilde et al (2014). "Genetics of cardiac arrhythmias." Heart 2005;91:1352–1358. doi: 10.1136/hrt.2004.046334 https://doi.org/10.1136/hrt.2004.046334
31. Jelle P.G. van der Ven et al (2019), Current outcomes and treatment of tetralogy of Fallot https://sci-hub.se/10.12688/f1000research.17174.1
32. Berul, C. I., et al. (2006). "Sudden death in congenital heart disease." Circulation, 114(2), 217-227.https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.105.592295???
33. E. Braunwald et al, 2007 Update to the ACC/AHA Guidelines for the Management of Patients With Unstable Angina and Non–ST-Segment Elevation Myocardial Infarction: Implications for Emergency Department Practice. Annals of Emergency Medicine, Volume 51, Issue 5, May 2008, Pages 591-606 https://www.sciencedirect.com/science/article/abs/pii/S0196064407015405
34. Jessup, M., & Brozena, S. (2003). "Heart failure." New England Journal of Medicine, 348(20), 2007-2018. https://www.nejm.org/doi/full/10.1056/NEJMra021498
35. Carabello, B. A. (2005). "Modern management of mitral stenosis." Circulation, 2005 Jul 19;112(3):432-7. https://pubmed.ncbi.nlm.nih.gov/16027271/
36. Cooper, L.T. et al. "Myocarditis."New England Journal of Medicine, 2009. https://www.nejm.org/doi/full/10.1056/NEJMra0800028
37. Klein, I., & Ojamaa, K. (2001). "Thyroid hormone and the cardiovascular system." New England Journal of Medicine, 344(7), 501-509. https://www.nejm.org/doi/full/10.1056/NEJM200102153440707
38. Haissaguerre, M., et al. (1998). "Sudden cardiac arrest associated with early repolarization." New England Journal of Medicine, 358(19), 2016-2023. https://www.nejm.org/doi/full/10.1056/NEJMoa071968
39. Batts, K., Ackermann, D., & Edwards, W. (1990). Postinfarction rupture of the left ventricular free wall: clinicopathologic correlates in 100 consecutive autopsy cases.. Human pathology, 21 5, 530-5. https://doi.org/10.1016/0046-8177(90)90010-3.
40. Shapira, I., Isakov, A., Burke, M., & Almog, C. (1987). Cardiac rupture in patients with acute myocardial infarction.. Chest, 92 2, 219 23. https://doi.org/10.1378/CHEST.92.2.219.
41. Lewis, A., Burchell, H., & Titus, J. (1969). Clinical and pathologic features of postinfarction cardiac rupture.. The American journal of cardiology, 23 1, 43-53. https://doi.org/10.1016/0002-9149(69)90240-9.
42. Tao, Z., Cavasin, M., Yang, F., Liu, Y., & Yang, X. (2004). Temporal changes in matrix metalloproteinase expression and inflammatory response associated with cardiac rupture after myocardial infarction in mice.. Life sciences, 74 12, 1561-72 . https://doi.org/10.1016/J.LFS.2003.09.042.
43. J. Ottosen et al (2012) Blunt cardiac injury, the american association for the surgery of trauma ; https://www.aast.org/resources-detail/blunt-cardiac-injury

*Corresponding Author: **Leila BELAROUSSI**

44. F. D. GIOGIO et al, homicide by heart attack? Legal Medicine 11 (2009) S531–S532

*Corresponding Author: **Leila BELAROUSSI**